

# Bacteriology of Symptomatic Adenoids in Children

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## Abstract

**Background:** Children with adenoid hypertrophy have been shown to harbor pathogenic bacteria in the nasopharynx despite antibiotics. Removal of the adenoid is associated with a reduction in the bacterial count. **Aims:** The study was done to determine the bacteriology of the adenoid tissue in chronic adenotonsillitis and adenoid hypertrophy, and determine the antibiotic sensitivity of potential pathogens. **Materials and Methods:** This is a descriptive study conducted on 100 patients aged between three and twelve years who underwent adenotonsillectomy/adenoidectomy. After adenoidectomy, the specimen along with the swab taken from the surface of the adenoid was sent for microbiological examination. After 48 and 96 hours, the microbial growth was identified and the antibiotic-sensitivity pattern of the isolate was studied. **Results:** Aerobic organisms grew in 93% of the specimens and anaerobic organisms in 68%, whereas 7% had no growth. The surface was predominated by commensals and the pathogens were mainly found in the core. The predominant pathogens were *Staphylococcus aureus*, *Streptococcus pneumoniae*, and *Enterococcus* species. The organisms were resistant to penicillin but showed sensitivity to co-amoxiclav and ciprofloxacin. Co-amoxiclav and ciprofloxacin should be considered as the first line of medical treatment for adenotonsillar diseases. **Conclusions:** Infection is the main cause of adenoid hypertrophy. Amoxicillin with potassium clavulanate and ciprofloxacin should be considered as the drugs of choice for all adenotonsillar diseases. Early and prompt treatment of adenoid hypertrophy with appropriate antibiotics will avoid unnecessary exposure to repeated antimicrobial therapy, thereby maintaining the beneficial effects of the normal adenoid flora.

**Keywords:** Adenoid, Adenoid hypertrophy, Antibiotic sensitivity, Microbial flora

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## Introduction

The adenoids are believed to play a role in several infectious and noninfectious upper airway disorders in children. Adenoidectomy is frequently performed to relieve recurrent ear infections and chronic adenoiditis associated with persistent ear effusions in children. These children are usually treated with multiple courses of antibiotics prior to the surgery; however, many continue to harbor pathogenic bacteria in their nasopharynx.

Various theories have been suggested to explain the persistence of these pathogenic organisms in

the nasopharynx. These include the appearance of penicillin-resistant alpha-hemolytic streptococci and increased numbers of beta-lactamase-producing organisms such as *Staphylococcus aureus* and some strains of *Haemophilus influenzae*. Removal of the adenoids is associated in many instances with a reduction of these pathogenic organisms.

The present study aims to ascertain the aerobic and anaerobic bacteriology on the surface and core of the adenoid tissue in children with chronic adenotonsillitis and adenoid hypertrophy causing nasal obstruction and ear manifestations which might help in the choice of appropriate antibiotics for treatment.

## Materials and Methods

Written informed consent was taken from the parents of the patients in the prescribed consent form. Approval was obtained from the local review board and the ethical committee. This descriptive study was conducted on 100 patients who underwent

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adenotonsillectomy/adenoidectomy for chronic adenotonsillitis or adenoid hypertrophy. Children in the age group between 3 and 12 years with chronic adenotonsillitis or symptoms due to adenoid hypertrophy were included in the study. They were divided into two groups. Children suffering from recurrent adenotonsillitis were included in group A. This group presented with persistent or recurrent mucopurulent nasal discharge, bilateral nasal obstruction, and recurrent throat pain. Group B included children who presented with persistent mouth breathing, snoring, nasal obstruction, or ear manifestations from adenoid hypertrophy but did not have nasal discharge or throat pain. Children who received antimicrobial therapy within one month prior to surgery were excluded from the study. Those with other local pathologies such as cleft palate and allergy were also excluded from the study.

All patients underwent routine blood investigations including hemoglobin, total count, differential count, erythrocyte sedimentation rate, bleeding time, clotting time, blood grouping, and crossmatching. A detailed ear, nose, and throat examination along with pure tone and impedance audiogram was done for all patients. The presence of adenoid hypertrophy was confirmed in both the groups by taking a soft tissue X-ray of the nasopharynx in the lateral view. Prior to surgery, blood was sent for culture with all aseptic precautions. All patients were operated under general anesthesia with orotracheal intubation. At the commencement of the surgery, a sterile swab was taken with a cotton-tipped applicator from the surface of the adenoid. Adenoidectomy was done by curettage method using adenoid curette and the specimen was immediately transported in normal saline to the microbiology laboratory in a sterile bottle along with the surface swab. The core adenoid tissue was excised from the deep surface of the adenoid specimen with a knife. The surface swab and the core adenoid tissue were plated onto aerobic and anaerobic media separately. The specimen was inoculated onto 5% sheep blood agar, chocolate agar, and MacConkey agar plates for the recovery of aerobic and facultative anaerobic organisms. The plates were incubated at 37°C aerobically (MacConkey) and under 5% carbon dioxide (5% sheep blood agar and chocolate agar plates) and examined after 24 and 48 hours of incubation. For anaerobes, the material was plated onto 5% sheep blood agar containing kanamycin or thioglycolate broth and then incubated in an anaerobic jar with gas-pak and then examined at 48 and 96 hours. The growth was identified by Gram stain and biochemical tests. The antibiotic-sensitivity pattern of the isolate was studied by modified Kirby-Bauer disc diffusion method and the antibiogram of the isolate determined. The specimen was also sent for histopathological examination.

## Statistical analysis

The results were analyzed and represented using bar charts, graphs, and tables.

## Results

Of the 100 children, 57 were males and 43 were females. Male-to-female (M:F) ratio was 1.32:1. There were 88 patients in group A of whom 67 had chronic adenotonsillitis, 15 had chronic adenotonsillitis with otitis media with effusion (OME), and six had chronic adenotonsillitis with chronic suppurative otitis media (CSOM). Group B included 12 patients of whom nine had chronic adenoid hypertrophy and three had chronic adenoid hypertrophy with OME [Table 1]. There was no difference in the pathogens isolated from the adenoid in chronic adenotonsillitis as compared with chronic adenoid hypertrophy alone. Majority of the patients presented with two to three years of prior symptoms. All our patients had complaints of mouth breathing and snoring mainly at night, observed by the parents. Fourteen children also had constant nasal obstruction and were mouth breathers during the day as well. Sore throat was present in 88 children, hearing loss in 24, ear discharge in six, and one child had epistaxis [Table 2].

## Tissue bacterial status

Aerobic organisms grew in 93% of the specimens and anaerobic organisms grew in 68% of the specimens, whereas 7% of the specimens had no growth of aerobes or anaerobes [Table 3]. The swab from the surface of the

**Table 1: Case distribution**

Division of Patients	Type of cases	No.	%
Group A	Chronic adenotonsillitis	67	67
	Chronic adenotonsillitis+OME	15	15
	Chronic adenotonsillitis+CSOM	6	6
Group B	Chronic adenoid hypertrophy	9	9
	Chronic adenoid hypertrophy+OME	3	3
Total		100	100

CSOM: Chronic suppurative otitis media; OME: Otitis media with effusion

**Table 2: Symptomatology**

Symptoms	No. of patients (total: 100)	(%)
Mouth breathing	100	100
Snoring	100	100
Nasal obstruction	14	14
Sore throat	88	88
Ear discharge	6	6
Hearing loss	24	24
Epistaxis	1	1

**Table 3: Tissue bacterial status**

Type of growth	No. of specimens		Total no. of specimens
	Group A	Group B	
Aerobic organisms	81	12	93
Anaerobic organisms	60	8	68
No growth	7	0	7
Total no. of specimens			100

adenoid revealed mainly commensals and some showed the same organisms as the core tissue. So, only the core tissue was considered for the study. The core bacterial flora was subjected to culture and sensitivity.

### Microbial profile of core adenoid tissue

Among the aerobic organisms, Gram-positive organisms like *Streptococcus pyogenes*, *Staphylococcus epidermidis*, *Streptococcus viridans*, *S. aureus*, *Streptococcus pneumoniae*, and *Enterococcus* species were seen. The Gram-negative organisms included *Pseudomonas*, *Klebsiella*, *Escherichia coli*, *Moraxella*, and *Haemophilus influenzae*. The anaerobic flora included *Peptostreptococcus*, *Prevotella*, *Fusobacterium*, and *Bacteroides*. All the adenoid samples sent for histopathological examination showed chronic inflammatory infiltrates including the culture-negative specimens (no growth) [Table 4].

### Antibiotic sensitivity of the isolates

The probable pathogens were subjected to antibiotic sensitivity and the antibiogram thus obtained showed the following results:

No single pathogen showed predominance over another. Of the 100 specimens cultured, there were 34 isolates of *S. aureus*, 39 isolates of *S. pneumoniae*, and 36 isolates of *Enterococcus* species. Antibiotic sensitivity was tested for commonly used drugs: Penicillin, ampicillin, amoxiclav, ciprofloxacin, ceftazidime, and ceftriaxone. None of the isolates of *S. aureus* and *Enterococcus* were sensitive to penicillin and ampicillin. In addition, enterococci species were resistant to third-generation cephalosporins like ceftazidime and ceftriaxone. Twenty-five *S. aureus* isolates were sensitive to ciprofloxacin, whereas 15 were sensitive to amoxiclav. *Enterococcus* species showed 28 isolates as sensitive to amoxiclav and 12 to ciprofloxacin. *Streptococcus* isolates showed varied but significant sensitivity to all the drugs mentioned. Twenty-eight isolates were sensitive to penicillin, ampicillin, and amoxiclav. Twenty-five isolates were sensitive to ciprofloxacin, 24 isolates were sensitive to ceftazidime, and 22 were sensitive to ceftriaxone. In the case of Gram-negative organisms, antibiotic sensitivity was tested for amoxiclav, amikacin, gentamicin, ceftriaxone, ciprofloxacin, and ceftazidime. Only one isolate of *Pseudomonas* was seen which was sensitive to amikacin.

**Table 4: Showing aerobic and anaerobic bacterial profile**

Aerobic and anaerobic organisms	Group A (88 patients)	Group B (12 patients)	Total (100 patients)
<i>Streptococcus pyogenes</i>	11	4	15
<i>Streptococcus viridans</i>	17	1	18
<i>Staphylococcus epidermidis</i>	2	0	2
<i>Staphylococcus aureus</i>	26	8	34
<i>Streptococcus pneumoniae</i>	34	5	39
<i>Enterococcus</i>	34	2	36
<i>Pseudomonas</i>	0	1	1
<i>Klebsiella</i>	6	0	6
<i>Escherichia coli</i>	2	0	2
<i>Haemophilus influenzae</i>	2	0	2
<i>Moraxella</i>	10	3	13
<i>Peptostreptococcus</i>	33	3	36
<i>Prevotella</i>	10	4	14
<i>Bacteroides</i>	23	1	24
<i>Fusobacterium</i>	3	0	3

Six isolates of *Klebsiella* were seen, all of which were sensitive to ciprofloxacin and ceftriaxone, whereas three were sensitive to amoxiclav, amikacin, gentamicin, and ceftazidime. Two isolates of *E. coli* grew which were sensitive to all the drugs except ciprofloxacin. Two isolates of *H. influenzae* also grew which were sensitive only to ciprofloxacin and ceftazidime [Table 5].

## Discussion

Adenotonsillar diseases pose a major problem in children and may require surgical intervention. It is estimated that worldwide 44–120 per 10,000 children (7.5–17.3% of all children) younger than 15 years undergo tonsillectomy and adenoidectomy. Apart from the associated surgical complications, the surgery also has psychological and financial impact on the patients and their families. Medical treatment for the eradication of infections appears to be a more suitable option.<sup>[1]</sup>

In our study, we aimed at studying the bacteriological pattern of surface and core adenoid tissue and the sensitivity of the probable pathogens to the common drugs used in the treatment of adenotonsillar diseases. In view of the morbidity and the burden of surgery that young children would be subjected to following adenoidectomy and also given the fact that adenoids would atrophy in due course of time by 10–12 years of age, we attempted to study whether a change in the

**Table 5: Antibiotic sensitivity pattern of isolates**

Organisms	No. of isolates	Antibiotics (sensitive)					
		P n (%)	A n (%)	Ac n (%)	Cf n (%)	Ce n (%)	Ci n (%)
<i>Staphylococcus aureus</i>	34	R	R	15 (44)	25 (73)	4 (11)	4 (11)
<i>Streptococcus pneumoniae</i>	39	28 (71)	28 (71)	28 (71)	25 (64)	24 (61)	22 (56)
<i>Enterococcus</i>	36	R	R	28 (77)	12 (33)	R	R
<i>Pseudomonas</i>	1	R	1 (100)	R	R	R	R
<i>Klebsiella</i>	6	3 (50)	3 (50)	3 (50)	6 (100)	3 (50)	6 (100)
<i>Escherichia coli</i>	2	2 (100)	2 (100)	2 (100)	R	2 (100)	2 (100)
<i>Haemophilus influenzae</i>	2	R	R	R	2 (100)	2 (100)	R

P: Penicillin; A: Ampicillin; Ac: Co-amoxiclav; Cf: Ciprofloxacin; Ce: Ceftazidime; G: Gentamycin; Ci: Ceftriaxone; Ak: Amikacin; R: Resistant

medical line of management could prove a better option in most of these cases. We divided the patients into two groups: One group who presented with recurrent adenotonsillitis and the other group who presented with persistent mouth breathing, snoring, nasal obstruction, and ear manifestations from adenoid hypertrophy. The purpose was to see if the organisms grown from the adenoid core differed in the two groups. We did not find much difference in the pathogens grown from adenoid tissue in chronic adenotonsillitis and those in adenoid hypertrophy; in our study, all 100 children had snoring. According to Anstead, 7 to 10% of the children between one and 10 years present with snoring with or without adenoid hypertrophy.<sup>[2]</sup> Behlfelt showed that 62.58% of the children with adenoid hypertrophy had mouth breathing during the day and 84.78% during nights.<sup>[3]</sup> In our study, all 100% were mouth breathers at night but 14% had mouth breathing during the day.

We found that the deep adenoid flora is polymicrobial in nature. Okur *et al.* found in their study that the organisms isolated from the adenoidal surface did not always show correspondence with the organisms isolated from the deep tissue specimens. Group A beta-hemolytic streptococci was the most commonly grown organism in the core of the adenoidal tissue and/or adenoidal surface culture, followed by *S. pneumoniae*, *S. aureus*, and *H. influenzae*. They isolated anaerobic bacteria from the core of the adenoid tissue but not from the surface of the adenoid.<sup>[4]</sup> In our study, we found that core and surface pathogens were corresponding, but in addition, the surface also had commensals. *S. pneumoniae* and *Enterococcus* species were the most common pathogens followed by *S. aureus* in our study. We recovered anaerobic bacteria both from the surface and core of the adenoid tissue. In both the groups, we found that Gram-positive organisms were predominant. The most frequently isolated pathogenic species were *S. aureus*, *Streptococcus* species especially *S. pneumoniae*, and *Enterococcus* species. *S. pyogenes*, *S. epidermidis*, and *S. viridans* were the commensal organisms grown. The Gram-negative organisms included *Pseudomonas*, *Klebsiella*, *E. coli*, *Moraxella*, and

*H. influenzae*, of which *Moraxella* is a commensal. The anaerobic flora included *Peptostreptococcus*, *Prevotella*, *Fusobacterium*, and *Bacteroides*. Our results are similar to Brook and Shah and Khalid *et al.*, who found a predominance of Gram-positive organisms with *S. aureus* being the most commonly isolated species, followed by *Streptococcus* species.<sup>[1,5]</sup> The Gram-negative organisms were grown to a lesser extent. Brook and Shah found several anaerobes in the adenoids of children including *Peptostreptococcus*, *Prevotella*, *Fusobacterium*, and *Bacteroides* which is similar to our study. Khalid *et al.* had no patients with anaerobes in their study. Taylan isolated *S. viridans*, *Neisseria* species, and *H. influenzae* from the surface of the adenoid and *S. viridans*, *Neisseria* species, and coagulase-negative staphylococci from core adenoid samples. When micro-organisms from the core and surface of the adenoid were compared, production of micro-organisms on the surface of the adenoid was statistically significant.<sup>[6]</sup>

In the Brook and Bethesda study, the aerobic organisms most frequently isolated were alpha- and gamma-hemolytic streptococci, *S. aureus*, beta-hemolytic streptococci (groups A, B, C, and F), *Haemophilus* species (*H. influenzae* type B and *Haemophilus parainfluenzae*), and pneumoniae. *H. influenzae* and *S. aureus* were more frequently isolated in children with chronic adenotonsillitis. The predominant anaerobic organisms were *Bacteroides* species, *Fusobacterium* species, anaerobic Gram-positive cocci, and *Veillonella parvula*, whereas *Bacteroides fragilis* was recovered only in children with chronic adenotonsillitis. In our study, we recovered *Bacteroides* species from children with chronic adenotonsillitis as well as those with chronic adenoid hypertrophy.<sup>[7]</sup>

In our study, all patients had stopped antibiotic treatment one month prior to surgery. The negative blood cultures prior to surgery indicate the absence of any other source of infection. The histopathological study of the core tissue showed the presence of chronic inflammatory infiltrates that suggests that chronic inflammation in



the adenoid tissue is probably the cause for adenoid hypertrophy. We studied the antibiotic sensitivity of commonly used drugs including penicillin, amoxicillin, amoxiclav, ciprofloxacin, ceftazidime, ceftriaxone, and specific antibiotics against Gram-negative organisms like amikacin, gentamicin, and so on. As drug resistance is not known in anaerobic organisms, antibiotic sensitivity was not done. All the Gram-negative aerobes except *Moraxella* were studied for antibiotic sensitivity as they are pathogens, whereas in the Gram-positive group, only those considered as probable pathogens were subjected to antibiotic sensitivity.

In our study, the aerobes, both Gram positive and Gram negative, showed resistance to the penicillin group of drugs, except for *S. pneumoniae* species, where 71% isolates were found sensitive to penicillin and ampicillin. According to I. Brook, bacteria such as *S. aureus* and Enterobacteriaceae are resistant to penicillin.<sup>[7]</sup> He found that other previously susceptible organisms such as *H. influenzae* and *Moraxella catarrhalis* had started to develop resistance to penicillin, like in our study.

Many bacteria are inherently insensitive to penicillin either due to the drug being unable to reach the target enzyme or its receptor maybe located deep under the lipoprotein barrier of the bacterial cell wall or due to low affinity of the receptors for the drug. The primary mechanism of acquired resistance is by the production of penicillinase or beta-lactamase. Brook found that the production of beta-lactamase by bacteria is responsible for resistance to penicillin. These bacteria are not only resistant to penicillin therapy but also render the other penicillin-susceptible bacteria resistant to penicillins by releasing free enzymes into their environment.<sup>[8]</sup> We found that with the addition of a beta-lactamase inhibitor like potassium clavulanate, the sensitivity to amoxicillin increased in both Gram-positive and Gram-negative organisms. Ciprofloxacin (quinolone group) is known to have a wide range of bacterial coverage and the most susceptible is the aerobic Gram-negative bacilli, especially Enterobacteriaceae and *Neisseria*. In our study, the Gram-positive aerobes showed good sensitivity (*S. aureus*: 71%, *S. pneumoniae*: 64%, *Enterococcus*: 33%), whereas the Gram-negative organisms (*Klebsiella* and *H. influenzae*) showed 100% sensitivity to ciprofloxacin. Ceftazidime (third-generation cephalosporin) has highly augmented activity against Gram-negative Enterobacteriaceae, with specific activity against *Pseudomonas*. It is less active against Gram-positive cocci like *S. aureus*. Our study also showed that Gram-negative bacteria had good sensitivity to ceftazidime, with *Klebsiella* being 50% sensitive and *E. coli* and *H. influenzae* being 100% sensitive to the drug, whereas Gram-positive bacteria had poor sensitivity to the drug. In comparison, the combination

of amoxicillin and potassium clavulanate had better coverage of Gram-positive as well as Gram-negative aerobes according to our study. Brook and Gober found that amoxicillin/clavulanate was superior to amoxicillin in achieving clinical cure (92 versus 64%). They also found that amoxicillin/clavulanate was superior in reducing the number of potential nasopharyngeal pathogens including *S. pneumoniae*.<sup>[9]</sup> The total number of potential pathogens were lower in those treated with amoxicillin/clavulanate or clindamycin.<sup>[10,11]</sup> In another study, Brook I. found that a 10-day therapy of co-amoxiclav reduced the bacterial load as well as the potential pathogens.<sup>[12]</sup> In our study also, we found that co-amoxiclav was more effective against the potential pathogens as compared to amoxicillin alone. Sclafani *et al.* demonstrated a significant reduction in the need for adenotonsillectomy after 30 days of therapy with amoxiclav compared with placebo in children with hypertrophied adenoids and tonsils.<sup>[13]</sup> The misuse of conventional antibiotics has resulted in the problem of drug resistance. The process of discovering newer antibiotics is a lengthy one and requires a lot of financial resources.<sup>[14]</sup> Hence, it is important to have a scientific basis while prescribing antibiotics and this article aims to standardize the use of proper antibiotics in adenoid infection through culture studies.

From this study, we summarize that 1) The adenoid bacterial flora is polymicrobial in nature. There is no difference in the pathogens isolated from the adenoid in chronic adenotonsillitis as compared with chronic adenoid hypertrophy alone; 2) The bacteria on the surface of the adenoid correspond to the core bacterial flora. Although the surface is predominated by commensals, the pathogens are mainly found in the core tissue, as the core pathogens are identical to those found on the surface; 3) The predominant pathogens are the Gram-positive aerobes such as *S. aureus*, *S. pneumoniae*, and *Enterococcus* species; 4) As all the specimens showed chronic inflammatory infiltrates on histopathological examination, we believe that infection is the main cause for adenoid hypertrophy; and 5) Our study shows that the pathogenic organisms are resistant to penicillins which are the first line of drugs in medical treatment, although they have good sensitivity to amoxicillin with potassium clavulanate and ciprofloxacin. The production of beta-lactamase by bacteria is responsible for resistance to penicillin. These bacteria are not only resistant to penicillin therapy, but also render the other penicillin-susceptible bacteria resistant to penicillins by releasing free enzymes into their environment. Therefore, we suggest that amoxicillin with potassium clavulanate and ciprofloxacin should be considered as the drugs of choice for all adenotonsillar diseases, thus reducing morbidity due to adenoid hypertrophy in early childhood.

In conclusion, early and prompt treatment of adenoid hypertrophy with appropriate antibiotics will avoid unnecessary exposure to repeated antimicrobial therapy, thereby maintaining the beneficial effects of the normal adenoid flora. This will prevent colonization of the adenoid by potential pathogens which make the adenoid the reservoir for sinonasal and middle ear infections.

## References

1. Khalid A, Al-Mazrou, Abdulaziz S, Al-Khattaf. Adherent Biofilms in adenotonsillar diseases in children. Arch Otolaryngol Head Neck Surg 2008;134:20-3.
2. Anstead M. Pediatric sleep disorders: New developments and evolving understanding. Curr Opin Pulm Med 2000;6:501-6.
3. Behlfelt K. Enlarged tonsils and the effect of tonsillectomy. Characteristics of the dentition and facial skeleton. Posture of the head, hyoid bone and tongue. Mode of breathing. Swed Dent J 1990;72:1-35.
4. Okur E, Aral M, Yildirim I, Kiliç Ve MA, Çıragil P. Bacteremia during adenoidectomy. Int J Pediatr Otorhinolaryngol 2002;66:149-53.
5. Brook I, Shah K. Bacteriology of adenoids and tonsils in children with recurrent adenotonsillitis. Ann Otol Rhinol Laryngol 2001;110:844-8.
6. Taylan I, Ozcan I, Mumcuoglu I, Baran I, Ozcan KM, Akdogan O, *et al.* Comparison of the surface and core bacteria in tonsillar and adenoid tissue with beta lactamase production. Indian J Otolaryngol Head Neck Surg 2011;63:223-8.
7. Brook I. Bethesda: Aerobic and anaerobic bacteriology of adenoids in children: Comparison between patients with chronic adenotonsillitis and adenoid hypertrophy. Laryngoscope 1981;91:377-82.
8. Brook I. The role of beta-lactamase-producing-bacteria in mixed infections. BMC Infect Dis 2009;9:202:1471-2334.
9. Brook I, Gober AE. Effect of amoxicillin and co-amoxiclav on the aerobic and anaerobic nasopharyngeal flora. J Antimicrob Chemother 2002;49:689-92.
10. Brook I, Shah K. Effect of amoxicillin with or without clavulanate on adenoid bacterial flora. J Antimicrob Chemother 2001;48:269-73.
11. Brook I, Shah K. Effect of amoxicillin or clindamycin on the adenoids bacterial flora Otolaryngol Head Neck Surg 2003;129:5-10.
12. Brook I. Effects of Antimicrobial therapy on microbial flora of adenoids. J Antimicrob Chemother 2003;51:1331-7.
13. Sclafani AP, Ginsburg J, Shah MK, Dolitsky JN. Treatment of symptomatic chronic adenotonsillar hypertrophy with amoxicillin/clavulanate potassium: Short and long term results. Pediatrics 1998;101:675-81.
14. Simi SP, Sarala N. Newer antibacterials in therapy and clinical trials. N Am J Med Sci 2012;4:537-47.

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